Office Action Summary	Application No.	Applicant(s)	
	10/540,864	MATSUZAWA ET AL.	
	Examiner	Art Unit	
	Daniel M. Sullivan	1636	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *04 June 2007*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-29* is/are pending in the application.

4a) Of the above claim(s) *1-11 and 15-22* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *12-14 and 23-29* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *06 February 2006* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☒ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *6/05, 2/06*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☒ Other: *See Continuation Sheet*.

Continuation of Attachment(s) 6). Other: sequence alignment US-10-540-864-1.rng .

DETAILED ACTION

This is the First Office Action on the Merits of the Application filed 27 June 2005 as the US national stage of international application PCT/JP03/16772 filed 25 December 2003, which claims benefit of Japanese patent application 2002-376589 filed 26 December 2002. The preliminary amendments filed 27 June 2005, 6 February 2006 and 4 June 2007 have been entered. Claims 1-22 were originally filed. Claims 12-14 were amended and claims 23-29 were added in the 4 June preliminary amendment. Claims 1-29 are pending.

Election/Restrictions

Applicant's election without traverse of Group II (claims 12-14 and newly added claims 23-29) in the reply filed on 4 June 2007 is acknowledged.

Claims 1-11 and 15-22 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected invention, there being no allowable generic or linking claim. Election was made **without** traverse in the 4 June reply.

Claim Amendments

The submission filed 4 June 2007 does not comply with the manner of making claim amendments set forth in 37 CFR § 1.121. Specifically, the markings to show changes made in claims 12-14 are not consistent with the amendments actually made relative to the previously entered claims. In the interest of compact prosecution, the claims have been examined as written in the 4 June claim set. However, any future submissions that do not fully comply with the requirements of 37 CFR § 1.121 will be considered non-compliant. For clarity, claims 12-14 are

set forth herein below with markings to show all changes made relative to the 27 June 2005 claims.

12. (currently amended) A screening method ~~of~~ for a compound which is capable of enhancing human adiponectin promoter activity or a salt thereof, which ~~comprising~~ comprises using ~~the~~ a transformant ~~according to claim 11~~ transformed with a recombinant plasmid DNA comprising DNA which consists of a promoter region having the nucleotide sequence of SEQ ID NO: 1 which comprises a regulatory sequence of a human adiponectin gene.

13. (currently amended) A screening method ~~of~~ for a preventive and/or therapeutic medicine for syndromes selected from syndrome X, metabolic syndrome, multiple risk factor syndrome, insulin resistance syndrome, deadly quartet, and visceral fat syndrome, which comprises using ~~the~~ a transformant ~~according to claim 11~~ transformed with a recombinant plasmid DNA comprising DNA which consists of a promoter region having the nucleotide sequence of SEQ ID NO: 1 which comprises a regulatory sequence of a human adiponectin gene.

14. (currently amended) The screening method according to claim 13, ~~which a disorder as a~~ wherein an etiology of the syndrome is diabetes, obesity, hypercholesterolemia, hyperlipoproteinemias, hyperlipidemia, arteriosclerosis, hypertonics, circulatory system disease, or polyphagies.

Claim Objections

Claims 24-29 are objected to under 37 CFR 1.75(c), as being of improper dependent form for failing to further limit the subject matter of a previous claim. Applicant is required to cancel the claim(s), or amend the claim(s) to place the claim(s) in proper dependent form, or rewrite the claim(s) in independent form.

The claims are directed to the method according to claims 12 or 13, wherein the regulatory sequence is a sequence containing PPRE, LRH-RE, SEQ ID NO: 2 and/or 3, or SEQ ID NO: 4. The DNA sequence of the base claim is limited to comprising a promoter region having the sequence of SEQ ID NO: 1. SEQ ID NO: 1 comprises SEQ ID NO: 2 (i.e., nucleotides 623-634 of SEQ ID NO: 1), SEQ ID NO: 3 (i.e., nucleotides 671-679 of SEQ ID NO: 1) and SEQ ID NO: 4 (i.e., nucleotides 623-679 of SEQ ID NO: 1). Therefore, by limiting the promoter region to comprising SEQ ID NO: 1, the claims are already limited to comprising SEQ ID NO: 2, 3 and 4 as recited in claims 26-29. Furthermore, SEQ ID NO: 2 is identified in the application as a PPRE (see especially Example 4, beginning on page 22) and SEQ ID NO: 3 is identified as a LRH-RE (see especially Example 5, beginning on page 23). Therefore, by limiting the promoter region to comprising SEQ ID NO: 1 the claims are also already limited to comprising a PPRE and LRH-RE as recited in claims 24 and 25. Consequently, claims 24-29 improperly fail to further limit the parent claims.

Claim 23 is objected to because of the following informalities: The phrase "comprising the regulatory sequence of human adiponectin gene can express" is grammatically incorrect. It is suggested that the phrase be amended to insert "the" between the words "of" and "human" and the phrase "can express" be deleted from the claim. Appropriate correction is required.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 13, 14 and 23-29 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a screening method for a compound which is capable of enhancing human adiponectin promoter activity, does not reasonably provide enablement for a screening method for a preventive and/or therapeutic medicine for syndromes selected from syndrome X, metabolic syndrome, multiple risk factor syndrome, insulin resistance syndrome, deadly quartet, and visceral fat syndrome. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and the invention commensurate in scope with these claims.

There are many factors to be considered when determining whether there is sufficient evidence to support a determination that a disclosure does not satisfy the enablement requirement and whether any necessary experimentation is "undue." These factors include, but are not limited to: (a) the nature of the invention; (b) the breadth of the claims; (c) the state of the prior art; (d) the amount of direction provided by the inventor; (e) the existence of working examples; (f) the relative skill of those in the art; (g) whether the quantity of experimentation needed to make or use the invention based on the content of the disclosure is "undue"; and (h) the level of predictability in the art (MPEP 2164.01 (a)).

Nature of the invention and Breadth of the claims: The claims are directed to a screening method for a preventive and/or therapeutic medicine for syndromes selected from syndrome X,

metabolic syndrome, multiple risk factor syndrome, insulin resistance syndrome, deadly quartet, and visceral fat syndrome, which comprises using a transformant comprising a regulatory sequence of a human adiponectin gene. Furthermore, claim 14 recites that the medicine has the capacity to prevent and/or treat any of the recited syndromes wherein the underlying cause of the syndrome can be any condition selected from diabetes, obesity, hypercholesterolemia, hyperlipoproteinemias, hyperlipidemia, arteriosclerosis, hypertonics, circulatory system disease, or polyphagies. As the claimed method is explicitly limited to identifying a medicine having the properties of a preventive and/or therapeutic medicine for syndromes selected from syndrome X, metabolic syndrome, multiple risk factor syndrome, insulin resistance syndrome, deadly quartet, and visceral fat syndrome, the disclosure must teach the skilled artisan how to practice the claimed method such that a medicine having the recited properties is obtained without undue experimentation.

Amount of direction provided by the inventor and existence of working examples: The instant application discloses the 5' regulatory sequence of the human adiponectin gene and the identification of certain regulatory elements within said 5' regulatory sequence. (See especially Examples 1-6.) The specification also teaches that the disclosed regulatory region could be operably linked to a reporter gene and induction of reporter gene expression by fetal bovine serum and pioglitazone could be detected. (Example 7.) Beyond this disclosure, the application asserts that adiponectin is an antidiabetic, antiarteriosclerotic, and antiobestic hormone, which is closely associated with the onset and progress of metabolic diseases based on an analysis of some prior art references. (See especially the paragraph bridging pages 1-2.) The application concludes that "[I]f a subject compound explored by using the above screening method or the

screening kit enhances the promoter activity of human adiponectin gene, it can increase the production and secretion of adiponectin in adipose tissue and thereby increase the plasma adiponectin concentration. Therefore, the compound can be used as a preventive and/or therapeutic medicine for metabolic disorder such as diabetes, obesity, hypercholesterolemia, hyperlipoproteinemia, hyperlipidemia, arteriosclerosis, hypertonia, circulatory system diseases, and hyperphagia, etc." (Page 11, lines 14-20.)

However, the application does not demonstrate that an agent capable of altering expression from the promoter construct disclosed in the instant application is capable of treating any of the syndromes recited in the instant claims or provide any direct evidence that agents identified by the claimed method would be capable of preventing and/or treating syndrome X, metabolic syndrome, multiple risk factor syndrome, insulin resistance syndrome, deadly quartet, and visceral fat syndrome.

State of the prior art and level of predictability in the art: The "predictability or lack thereof" in the art refers to the ability of one skilled in the art to extrapolate the disclosed or known results to the claimed invention. If one skilled in the art can readily anticipate the effect of a change within the subject matter to which the claimed invention pertains, then there is predictability in the art. On the other hand, if one skilled in the art cannot readily anticipate the effect of a change within the subject matter to which that claimed invention pertains, then there is lack of predictability in the art. Accordingly, what is known in the art provides evidence as to the question of predictability.

The physiological art is recognized as unpredictable. (MPEP 2164.03.) In cases involving predictable factors, such as mechanical or electrical elements, a single embodiment provides

broad enablement in the sense that, once imagined, other embodiments can be made without difficulty and their performance characteristics predicted by resort to known scientific laws. In cases involving unpredictable factors, such as most chemical reactions and physiological activity, the scope of enablement obviously varies inversely with the degree of unpredictability of the factors involved.

The prior art, like the instant specification, teaches that adiponectin is an important adipocyte-derived regulatory factor involved in a variety of metabolic processes and a potential therapeutic agent. (See especially Díez et al. (2003) *Eur. J. Endocrinol.* 148:293-300, which corroborates the teachings of the specification with regard to the effects of adiponectin deficiency and administration of exogenous adiponectin in mammals.) However, with regard to application of adiponectin to the treatment of any condition, Díez et al. teaches, "Testing these hypotheses [that adiponectin can be used to treat various conditions] is a challenge for future clinical research. Further investigations in patients with the above-mentioned states and other hypoadiponectinemic situations are required to clarify these aspects of the potential therapeutic applications of this fascinating adipocytokine." (Page 298, final two sentences.) Thus, the art teaches that, at the time the instant application was filed, adiponectin had not been established as an effective in the treatment or prevention of any condition.

In addition, the claimed method purports to use regulation of an adiponectin reporter construct as a surrogate endpoint for therapeutic or prophylactic efficacy in the treatment of a variety of syndromes. However, the art generally teaches that before a putative biomarker can be used as a surrogate endpoint it must be validated as such. Wagner (2002) *Dis. Markers* 18:41-46 acknowledges in the Abstract, "Putative biomarkers are typically identified because of a

relationship to known or hypothetical steps in a pathophysiologic cascade. Biomarker discovery can also be effected by expression profiling experiment using a variety of array technologies and related methods." However, Wagner cautions, "A rational basis for recommending the use of a putative biomarker does not guarantee the utility of the biomarker or its qualification as a surrogate endpoint" (paragraph bridging the left and right columns on page 43) and "Biomarkers require validation in most circumstances" (paragraph bridging pages 43-44).

Frank *et al.* (2003) *Nature Rev.* 2:566-580 concurs, stating, "The standard concepts of test-re-test reliability and validity apply with equal force to clinical biomarkers as they do in any assay system" and, "The work required to establish the reliability and validity of a new biomarker should not be underestimated in general, and in particular needs of planning for each combination of clinical indication and mechanism of action" (paragraph bridging the left and right columns on page 568). Feng *et al.* (2004) *Pharmacogenomics* 5:709-719 teaches, "The development and validation of clinically useful biomarkers from high-dimensional genomic and proteomic information pose great research challenges. Present bottle necks include: that few of the biomarkers showing promise in initial discovery were found to warrant subsequent validation...A molecular profiling approach, although promising, has a high chance of yielding biased results and overfitted models" (Abstract).

Viewed as a whole, the art clearly teaches that the utility of a putative biomarker as a surrogate endpoint for any disease state is unpredictable and must be validated.

Relative skill of those in the art and quantity of experimentation needed to make or use the invention: Although the relative level of skill in the art is high, the skilled artisan would not be able to use the claimed method to identify an agent useful as a preventive and/or therapeutic

medicine for syndromes selected from syndrome X, metabolic syndrome, multiple risk factor syndrome, insulin resistance syndrome, deadly quartet, and visceral fat syndrome without first having to engage in undue experimentation to establish that the method is a valid marker for therapeutic efficacy in the treatment of each of the recited syndromes. The art clearly establishes that putative biomarkers must be validated and that "few of the biomarkers showing promise in initial discovery were found to warrant subsequent validation" (Feng *et al.*, *Id.*). Furthermore, the art teaches that the therapeutic or prophylactic efficacy of adiponectin had not yet been verified for any of the syndromes recited in the claims at the time the application was filed.

Given this high degree of unpredictability and the absence of any evidence to establish that expression in the transformant of the claims gene is a valid surrogate endpoint for therapeutic efficacy in the treatment of the syndromes recited in the claims, the basic premise underlying the claimed invention is no more than a theoretical possibility. This is not sufficient to meet the enablement requirement of 35 USC §112, first paragraph.

> Patent protection is granted in return for an enabling disclosure of an invention, not for vague intimations of general ideas that may or may not be workable. *See Brenner v. Manson*, 383 U.S. 519, 536, 148 USPQ 689, 696 (1966) (stating, in context of the utility requirement, that 'a patent is not a hunting license. It is not a reward for the search, but compensation for its successful conclusion.') Tossing out the mere germ of an idea does not constitute enabling disclosure. While every aspect of a generic claim certainly need not have been carried out by an inventor, or exemplified in the specification, reasonable detail must be provided in order to enable members of the public to understand and carry out the invention. *Genentech Inc. v. Novo* Nordisk A/S (CA FC) 42 USPQ2d 1001, 1005.

In view of the foregoing, it would require undue experimentation to practice the invention claimed. Therefore, the claims are properly rejected under 35 USC §112, first paragraph, as lacking an enabling disclosure.

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 12-14 and 23-29 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The claims are indefinite in that they are directed to a process but fail to set forth any process steps. Instead, the claims merely recite an outcome and that the outcome is achieved using a specific transformant. Attempts to claim a process without setting forth any steps involved in the process generally raises an issue of indefiniteness under 35 U.S.C. 112, second paragraph. For example, a claim which read: "A process for using monoclonal antibodies of claim 4 to isolate and purify human fibroblast interferon." was held to be indefinite because it merely recites a use without any active, positive steps delimiting how this use is actually practiced. *Ex parte Erlich*, 3 USPQ2d 1011 (Bd. Pat. App. & Inter. 1986). See MPEP §2173.05(q).

Claim Rejections - 35 USC § 103

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later

invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 12-14 and 23-29 are rejected under 35 U.S.C. 103(a) as being unpatentable over Dufaure-Gare et al. WO 00/26363 (made of record in the IDS filed 27 June 2005).

The claims are directed to a screening method for a compound which is capable of enhancing human adiponectin promoter activity which comprises using a transformant transformed with a recombinant plasmid DNA comprising DNA which consists of a promoter region having the nucleotide sequence of SEQ ID NO: 1 which comprises a regulatory sequence of a human adiponectin gene.

It is first noted that the claims recite that the transformant is transformed with a plasmid DNA comprising DNA which consists of a promoter region having the nucleotide sequence of SEQ ID NO: 1. Although the claim uses the closed language "consists of" the transitional phrase is viewed as applying only to the limitation "promoter region" while the open transition "having" applies to the nucleotide sequence. Therefore, the claims are viewed as reading on any transformant transformed with a DNA that comprises SEQ ID NO: 1 within a "promoter region". In other words, the plasmid DNA of the claims is not limited to comprising a promoter region that consists of SEQ ID NO: 1.

In the section entitled, "Method For Screening Ligands That Modulate The Expression Of The APMl Gene", (beginning at page 102) Dufaure-Gare et al. teaches, "[A] method for screening substances or molecules that are able to increase, or in contrast to decrease, the level of expression of the APMI gene. Such a method may allow the one skilled in the art to select substances exerting a regulating effect on the expression level of the APMl gene and which may

be useful as active ingredients included in pharmaceutical compositions for treating patients suffering from deficiencies in the regulation of expression of the APMl gene, particularly patients suffering from obesity." (Page 103, lines 20-25.) Dufaure-Gare et al. further teaches that the method comprises providing a recombinant cell host containing a nucleic acid, wherein said nucleic acid comprises a nucleotide sequence of SEQ ID NO: 2 (i.e., the promoter sequence of the human adiponectin gene; referred to by Dufaure-Gare et al. as the APM1 gene) located upstream of a polynucleotide encoding a detectable protein. (See especially page 103, lines 28-30.)

SEQ ID NO: 2 of Dufaure-Gare et al. comprises the nucleotide sequence from 1 to 908 of the instant SEQ ID NO: 1. (See especially the sequence alignment US-10-540-864-1.rng mailed herewith.) Thus, Dufaure-Gare et al. teaches all of the elements of the method of the instant claim 12 except that Dufaure-Gare et al. fails to explicitly teach that the construct used in the method also comprises nucleotides 909-921 of the instant SEQ ID NO: 1. However, Dufaure-Gare et al. does go on to teach an embodiment of the screening method wherein the method is practiced using a host cell comprising the 5' UTR of the adiponectin cDNA, which is set forth as SEQ ID NO: 5 and nucleotides 1-13 of SEQ ID NO: 5 are identical to nucleotides 909-921 of the instant SEQ ID NO: 1. (See especially page l04, lines 15-32 and the SEQ ID NO: 5 sequence in the sequence listing.) Furthermore, Dufaure-Gare et al. teaches a specific embodiment wherein the 5' UTR sequence comprising SEQ ID NO: 5 includes a promoter sequence which is endogenous with respect to the APM1 5' UTR sequence (i.e., the sequence set forth as SEQ ID NO: 2). See especially page 104, lines 24-27. Still further, a review of the complete APM1 gene sequence, which is set forth in Dufaure-Gare et al. as SEQ ID NO: 1, reveals that SEQ ID NO: 2

(corresponding to nucleotides 1 to 908 of the instant SEQ ID NO: 1) is contiguous with the SEQ ID NO: 5 (containing nucleotides 909-921 of the instant SEQ ID NO: 1) in the native gene. (See the junction at nucleotides 4811-4812 of the Dufaure-Gare et al. SEQ ID NO: 1 sequence.)

It would have been obvious to one of ordinary skill in the art at the time the invention was made to practice the screening method of Dufaure-Gare et al. using a nucleic acid promoter comprising the entirety of the SEQ ID NO: 1 sequence. As described above, Dufaure-Gare et al. explicitly teaches practicing the method using sequence comprising nucleotides 1-908 of the instant SEQ ID NO: 1 and using nucleotides 909-921 of the instant SEQ ID NO: 1, that these sequences can be used together and that in the native gene nucleotides 1-908 of the instant SEQ ID NO: 1 are contiguous with nucleotides 909-921 of the instant SEQ ID NO: 1. One would be motivated to assemble the fragments as they are found in the native gene and the instant SEQ ID NO: 1 in order to preserve the natural configuration of the promoter/exon structure as it is found in the nature. Absent evidence to the contrary, one would have a reasonable expectation of success in combining the sequences because it would preserve the native structure of the naturally occurring functional gene.

In view of the foregoing, the invention of independent claim 12, as a whole, would have been obvious to one of ordinary skill in the art at the time the invention was made. With regard to claims 13 and 14, which are directed to the same process as the instant claim 12 and further reciting that the method screens for a preventive and/or therapeutic medicine for syndromes selected from syndrome X, metabolic syndrome, multiple risk factor syndrome, insulin resistance syndrome, deadly quartet, and visceral fat syndrome which result from any of a variety of etiologies, as the screening process disclosed in Dufaure-Gare et al. is the same as the process

claimed, it is presumed, absent evidence to the contrary, that the properties of the agents identified thereby are the same as the properties of the agent identified by the claimed method.

Claim 23 is directed to the method of claims 12 or 13 wherein the plasmid DNA is capable of expressing a structural gene under the control of the promoter region. As Dufaure-Gare et al. teaches a plasmid DNA comprising a functional promoter, the plasmid DNA of Dufaure-Gare et al. would be capable of expressing a structural gene. Therefore, the method of claim 23 would also be obvious over the teachings of Dufaure-Gare et al. for the reasons stated above.

Finally, as described herein above, the instant SEQ ID NO: 1 comprises the instant SEQ ID NO: 2-4, which comprise PPRE and LRH-RE sequences. Therefore, the method of Dufaure-Gare et al., which uses a promoter region comprising SEQ ID NO: 1 meets the limitations of the instant claims 24-29.

In view of the foregoing, the invention of claims 12-14 and 23-29, as a whole, would have been obvious to one of ordinary skill in the art at the time the invention was made. Therefore, the claims are properly rejected under 35 USC § 103(a) as obvious over the art.

Conclusion

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Daniel M. Sullivan whose telephone number is 571-272-0779. The examiner can normally be reached on Monday through Friday 6:30-3:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Joseph Woitach, Ph.D. can be reached on 571-272-0739. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Daniel M Sullivan/
Primary Examiner
Art Unit 1636